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A CLINIC GIVEN AT THE PETER BENT BRIGHAM HOSPITAL TO STUDENTS OF THE HARVARD MEDICAL SCHOOL

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Boston

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I HAVE one case (Med. No. 7527) to show this morning. This patient is one who came into the hospital the first time on September 24, and stayed until October 10, of this year, and then came back on November 9. He is thirty-nine years of age. He came in on account of soreness in his chest and abdomen, cough and hoarseness. His family history is negative. He has been married for thirteen years. He has one child, living and well. His wife has had no miscarriages. His habits are good. His occupation is that of a stationary steam engineer, and he works eight hours daily in a rather warm room. He has been at that work for twenty years. In his past history there are very few acute infectious diseases of any kind. He had measles and mumps when about fourteen years of age. He had two attacks of tonsillitis about eighteen years ago. There has been no recurrence of the tonsillitis. All of the other acute infectious diseases were asked about, and there is no history of them. His average weight has been about one hundred and forty-nine pounds, and he thinks that he has lost six or seven pounds during the past few weeks; that is prior to his first admission. About one and one-half years ago he quite suddenly had a sensation as though something was in his œsophagus that he could not swallow. That persisted for two or three weeks, and following that he had some soreness over his left chest. That evening he ate a pretty hearty dinner of spare ribs and cabbage. During the night he suffered from intense burning about the heart, which disappeared the next day, but the soreness then extended to the other side of his chest and to both sides of his abdomen. Since then he appears very often to have had soreness in his chest and soreness in his abdomen. That has not been noticeable all the time, but

has been present much of the time. There was no acute pain associated at that time with this soreness. Last May the symptoms began to get somewhat worse, and he stopped work in June. Since May he has been rather hoarse, had a dry cough and raised a little thin white sputum. During these various attacks, and especially for a period of a month or so before he came into the hospital, he had a slight afternoon fever, not very much, a degree or a degree and one-half. He has never raised any blood when he coughed, never had any night sweats during this period. Sometimes he had a little œdema about one or the other of his eyes in the morning, with a little itching in his eye. That apparently appeared and disappeared in attacks of short duration.

When he came into the hospital on his first admission and during his period in the hospital he ran a slight fever; only once did it reach a point nearly to 101. You notice that his pulse was a little more rapid than normal, but it was not a rapid pulse. Respiration was about normal. The curve of his temperature during his first admission is shown a little bit better in the four-hour chart, which brings out that variation during the day of about 2 degrees, with a period just before he left the hospital, when the temperature was more stationary, at about a level of 99.5; that is, it did not fluctuate, but he still continued to have a little fever. When he left the hospital he felt a good deal better than when he came in. The pain that he had in his chest and abdomen disappeared very considerably before he left, and after he left he did not have any pain until two weeks before his return to the hospital, when he had a sudden, sharp pain in the right side, which was made worse by breathing. This pain caused a certain amount of irritation, and the irritation was accompanied by cough. The cough was very painful on account of the stitch in his side. He remained at home for several days, keeping perfectly quiet. He had been going to the out-door department before that from time to time, but he felt so miserable with the pain in his side and heart that he did not go to the out-door department. When he did come in, his side was strapped, and this strapping gave him some relief.

Just before he came to the hospital the second time he noticed that his left arm about his elbow and his left wrist were somewhat swollen, and that when he moved his arm there was a creaking sensa-

tion. When he came to the out-door department there was swelling over the back of his hand, and as he moved the hand you could get distinctly fine crepitation. You could get the same thing just above his elbow. Since he has been in the hospital this time he has run just the same type of temperature curve. He has not been free from fever. The highest fever that he has had has been just over 100.5°. Nearly every afternoon the temperature goes up to 100. The fluctuations in the four-hour chart are from 1.5° to 2° during the twenty-four hours.

When he was in the hospital the first time on physical examination we made out friction rubs at the bases of both lungs, rather coarse and very distinct. We made out a friction rub over the gall-bladder region; as it moved you could get the sensation of crepitation with your fingers. If you listened you could get the distinct sound of a friction rub. His heart was enlarged to percussion when he came in the first time. Doctor Wearn percussed his left border 14 cm. to the left of the mid-sternal line and the right border 3 cm. No thrills were felt and no murmurs were heard. That was on September 24, and the next day, going over him, I made out the same size for the heart—14 cm. to the left and 3 cm. to the right. At this time the heart sounds were rather distant, but otherwise normal; that is, there were no murmurs. There was slight dulness over the right back on percussion, slightly decreased voice and breath sounds and decreased tactile fremitus. In the back and low down in the lower left axilla, a fine friction rub was heard, which largely disappeared after deep breathing. Over the lower portion of the right lung in front, along the costal margin, a coarser friction rub was heard. Those were the physical findings, and there was very little change during his stay in the hospital the first time. Sometimes these friction rubs were heard, and at other times they were not made out, so that they changed from day to day, though, usually, you could make out a friction rub. When he came in he had a very considerable secondary anæmia and had a little œdema, as we have already said. His hæmoglobin was 55 per cent. and red cells 2,616,000. There was slight irregularity in the red cells, but, essentially, the picture was that of secondary anæmia. As he had this secondary anæmia and ran a slight temperature, he was transfused, to bring up his hæmoglobin, to see if that would not improve his condition. On October

2 he received 600 Cc. of blood from an appropriate donor. He had no chill nor any reaction following transfusion. After that he felt very much better, his symptoms largely disappeared, and his general condition was very much improved.

Then when he came back for the second time, which was on November 9, the examination of his lungs showed the same condition that we found before—some dulness in the bases and some sounds suggesting râles, other sounds suggesting a friction rub, and in places the friction rub was of a coarse, leathery quality. Then there was a friction rub heard over his liver, beneath the right costal margin. The edge of the liver could be felt below the costal margin. The percussion of the heart gave the same figures, 14 cm. to the left and 3.5 cm. to the right, those being the figures of Doctor Wearn on November 9. The next day, on the ward visit, I noticed the friction rubs in the lower part of his chest and in the upper part of his abodmen—the right upper quadrant. I have already spoken of the feeling of crepitation that you could get when he moved his right wrist and elbow, and somewhat the same sounds were obtained in the right axilla by rubbing the muscles and subcutaneous tissue against each other. When he came in this second time he had the same type of anæmia which he had in the first admission. His blood had gone up somewhat with transfusion, but, apparently, had dropped again, as he had a hæmoglobin of 50 per cent. and a red count of 2,270,000 on his second admission. In both admissions he had a rather low leucocyte count. In the second admission, when he first came in, the count was 4400, on the 17th, 6600, and on the 24th, which was last Saturday, he had a count of 6800. There was nothing abnormal about the blood from the point of view of structure of the red cells or white cells. He has had a little cough, as described in the history, with scant amount of sputum coughed up. That has been examined repeatedly and no acid-fast organisms have been found. He had a blood culture made on November 13, and it was negative up to the 24th, so that he has a negative blood culture. His Wassermann reaction is negative. His phthalein examination is normal. His urine shows an occasional cast, a slight trace of albumen, and his stools are negative.

Since he has been in this time, apparently, there has been some slight increase in the heart size as made out by percussion, so that

on November 22 it was percussed out 4.5 cm. to the right and 16 cm. to the left. You remember it had been running 3 cm. to the right and 14 cm. to the left. It seemed at this time as if the left border of cardiac dulness was out considerably beyond the point at which you could feel any cardiac impulse. He never had a very marked cardiac impulse—a rather faint apex beat—and this could be felt not more than 13.5 cm. to the left, whereas the dulness was percussed further out. The heart sounds were as they had been in the past, rather distant and muffled, and there were no murmurs. There was no pulsation beyond the apex pulsation made out over the precordium. There was no systolic retraction anywhere. Both times in the hospital observations were made to see if there were any signs of retraction of the lower ribs on the left, so-called Broadbent's sign, but there was no evidence on any of these examinations of the presence of that sign.

At present he looks pretty comfortable. He does not look very sick and that is about the way he has looked since he has been in the various admissions. He has a little pallor probably. These marks on the chest are Dr. Wearn's percussion marks of yesterday. Correspondingly I made the right a little further out than Dr. Wearn did yesterday and the left a little further out. Our percussion notes have been running on the right at 3 to 4 and I think there was one at 5.5 as the maximum which would take it out here. That was, I think, my percussion note. Most of them have run out to where the right border is to-day, just about in this line—3 to 4 cm. out. The furthest out that his left has been is 16. That was on Saturday and his apex was felt here 13.5 cm. out, so that to-day the border is very distinctly inside where it was on Saturday. We also located his apex by auscultation and made it corresponding to about the point where we had located it by palpation. At present his apex is just inside where I put that dotted line by palpation, and by auscultation you get maximum sounds in the bell of the stethoscope just when the outer border of the stethoscope is just about where the percussion line is. So that the apex beat is now located in this point just a little inside of the nipple line. Apparently you can feel his liver edge but in that position propped up, it is not very satisfactory to get abdominal palpation and we won't go into the question in any detail.

Then we have had a number of X-ray plates of his chest. The striking thing when he first came into the Out-Door Department

before he came into the hospital, was that on September 19 he had that large globular heart. When he came into the Out-Door Department with that large heart he gave no history of any cardiac symptoms. You note in the history that I have given you there is none of the ordinary type of cardiac dyspnœa. There is no definite cardiac œdema, but when he came in he had this large globular heart without any obvious dyspnœa, without any œdema, without any cyanosis and with a pulse of normal rate. His pulse was not very much above normal at any time during that first stay in the hospital.

Here is the plate taken after he came into the ward on September 26. Here is a plate taken a little bit later, in this admission, on November 21. Here is a fourth plate, taken this morning. In the first place, in this first plate, except for the enlargement of the heart, there is practically nothing that is abnormal. The apices seem clear and the pulmonary tissue seems clear. There is a very definite angle here between the diaphragm and the ribs indicating that there is no fluid at that point. This diaphragm is a trifle higher and is somewhat more than usually dome shaped; that is, pushed a little high. On the other side, in just the same way, the phrenico-costal angle is clear, but there is a little haziness down in this portion of the plate, so essentially there is nothing in the lungs except down in this point there is a little more density than on the other side and more than is normal. A little bit later there is very little if any change in the general appearance of the heart shadow. There is no change in the size but this denser process along in this side is possibly a little more evident than on the other side. There is no change in the diaphragm. You notice there is a good deal more mottling here in the upper and middle part of the lungs in that plate than in this plate. That may not be due to difference in the pulmonary condition but due to difference in the time of exposure. A short exposure gives you more normal pulmonary mottling and a longer exposure gives you less, so I think in these plates there is no especial pulmonary change.

Here is a plate taken in September and there is one taken on November 21, which shows the heart shadow not so distinctly outlined, but you notice it out at this point, and then you see a line coming down obliquely here, starting at about the middle of the heart shadow and running out to the angle there between the ribs

and the diaphragm probably, though we cannot see in this plate. We can see the diaphragm distinctly here, which is about at the same level, but I cannot make out the diaphragm on that side. On the other side there is a little filling out of the angle. That air bubble in the stomach locates fairly well where the diaphragm is at that point. The left border of the heart cannot be made out with any assurance at all in this picture. We rather thought it ran where I am running my finger now. Of course, on that side the shadow is denser but here the shadow is more suggestive of pulmonary tissue as if this were the right border of the heart, here an area of increased pulmonary density and we percussed out in the back an area of dulness that corresponded fairly well with that outline.

Here is the plate taken this morning. You see this portion here of density has disappeared and you can see the diaphragm again quite distinctly at this point. The left border of the heart is about the same. If you come back and study the upper part of the heart you notice in these two plates a little incurving which represents the point at which the arch of the aorta comes down, a little curving out there, a distinct angle at this point and a large curve to the left border of the heart. That corresponds to the left ventricle, this to the left auricular appendage and that to the arch of the aorta. It is seen somewhat at a little different angle in this plate but you notice it is a distinct angle. When you come to this plate you notice that up at this point it is distinctly filled out and there is straightening out of the edge. Here is the arch of the aorta and you see there is that shadow but it is definitely a straight line, continuous with the arch of the aorta and coming down at that point to a slight angle and then rounding out. That oblique line has considerably disappeared in the plate of this morning, giving more of a curve in the arch of the aorta and then rounding out. This particular plate shows quite well the arch at that point and a straightening out of the angle.

Yesterday Dr. Wearn tapped his pericardium, going in at the point here where you can see the mark, which was just about where we had located the apex beat the day before and well inside the left border of cardiac dulness. Dr. Wearn made the dulness out further as if it had increased over night or as if he had percussed more delicately or differently from the way I did on Saturday.

(To Dr. Wearn:) "Did you locate the apex?"

Dr. Wearn: "I did not. I had him lying on the left side."

"When you percussed he was lying on that side—with that side down, so you could not feel the apex."

We went in at that point, which was presumably within the area of cardiac dulness, so as to just graze the apex. That is what we were attempting to do. We went in to see if there was any pericardial fluid, it being suggested by this long history and gradual enlargement in the size of the heart, slight temperature, etc., the question being as to whether he had a pericardial condition with a thickened pericardium and adhesions or whether he had pericardial effusion. We rather thought right along that he probably had a change in his pericardium with thickening and no fluid, but in the last note the signs were distinctly of an area of dulness beyond the area of apex impulse which always suggests fluid, but it does not necessarily point to fluid because sometimes from a thickened pericardium you will get the same thing. This straightening out of the line suggested fluid filling the pericardial sac, the pericardial sac being more or less of a bag, and distending from below upward as the fluid accumulates, it turns out nearly at this point and gives a straight line as the fluid accumulates. Dr. Wearn got 690 Cc. of fluid.

This (demonstration of specimen) looks very much like pleural fluid and that came from the pericardial sac. That fluid contains 310 cells per cubic centimeter of which 90 per cent. are mononuclears and 10 per cent. polymorphonuclears. It had a specific gravity of 1024. Clots have been examined and no tubercle bacilli were found in the clots. That is, we treated that pericardial fluid just as we treat pleural fluid. We examined the clots to see if any tubercle bacilli were present. Very frequently they are caught in clumps in the clotting and it is easier to find the organisms sometimes in clots than in free fluid. We also did as we do with pleural fluid, we injected some of it into the peritoneal cavity of a guinea pig to see if there is any evidence of tuberculosis as the cause of the fluid. I may say that we found no evidence of it in the examination of the lungs nor did we find any evidence of it in the examination of such sputum as he had.

That is a case, of course, of pericardial effusion. We have gotten 690 Cc. of fluid out of the pericardium, and that is all that could be obtained. He did not have any signs of embarrassment of his circulation as the result of that fluid in his pericardium. His pulse has not

been higher than 100 in the last few days. Some of the counts have been down to 86 and 87. The points of interest apart from the question of diagnosis are the etiology and question as to how long he has had this effusion and as to how much of this rounding out and enlargement of the shadow is due to pericardial effusion. You can see I think from these plates that without any question in the last group there has been a very definite enlargement of the heart shadow and following tapping, though with the position of the diaphragm and some obscuring here of the angle you cannot make out so distinctly the two borders of the heart, it does not look as if this shadow here is very much smaller, even though 600 Cc. of fluid have been removed. The fluid was removed yesterday and the X-ray was taken this morning, so it is possible that some of the fluid has reaccumulated.

At no time in our examination of his heart have we found any signs of pericardial friction. That is, while he has been under observation we have never had any signs of pericardial exudate with little or no fluid—the two surfaces coming together with a little pericardial friction rub. He has never had a pericardial friction rub. We have had a friction rub in the pleural cavity and a friction rub over the liver and gall bladder. We have had a friction rub in the tendon sheaths about his wrist and elbow. That is the significance of the fine crepitation that we got in moving the back of his hand up and down and moving his elbow joint. He has run this slight temperature.

When he was in the hospital the first time the abnormal things made out were these friction rubs and this enlargement of the heart. We could not make any diagnosis as to the cause of it, so we sent him out, having made the diagnosis of subacute fibrinous pleurisy, hypertrophy of the heart and secondary anæmia, we did not make any definite diagnosis of any pericardial lesion though we were a little inclined to think that cardiac enlargement without heart murmurs and without a history of rheumatism was probably due to pericardial thickening; that is, adherent pericardium, though he did not give any evident physical signs of such a change.

Before he came to the hospital, for a period of several weeks, and in the interval between the two admissions, his temperature was taken regularly by his sister, who happens to be a trained nurse, and all during that period he was running some kind of slight fever, and that fever was increased by exertion and exercise. If he kept per-

fectly quiet, stayed in the house and did not move around very much, the temperature went up a degree or a degree and one-half. If he took a walk, or any exercise, instead of going up a degree, it went up two and one-half degrees every afternoon. That is somewhat suggestive of the type of temperature that we get in pulmonary tuberculosis. The underlying thought that we had during his stay in the hospital the first time was that he did have a tuberculous focus somewhere, but we could not locate it. Though we did not get any signs of pulmonary tuberculosis, though we did not get any signs of pleurisy with effusion, no signs of tuberculous peritonitis, no signs of tuberculosis in any local glands or any of the joints, we still had the feeling that with that kind of temperature, with an evidently run-down condition and secondary anæmia, that the whole picture was very strongly suggestive of some tuberculous focus. We speak of those things as latent tuberculous foci. I might say, in that connection, that there was no evidence in the urine of any tuberculous process. There was no pus, frequency, hæmaturia, etc., so there was no evidence of a tuberculous focus in the kidney, which, of course, is a fairly frequent place for tuberculosis to develop and sometimes gives very few symptoms.

When he came back he continued to run just the same kind of temperature, evidently not having gotten any better nor, apparently, any worse. He came to the out-door department the last time because he had this pain in his side, which bothered him. I saw him out there one day. He had not gotten any change in condition from the point of view of getting any worse; but from the point of view of having a man running a temperature, slight but persistent, for which we had not found a cause, and as we did not seem to be getting very far on observing him in the out-door department, I sent him in for observation, and hoped that in the hospital we would clear up the diagnosis this time. His condition did not change much during that period of observation, and he was not sent back to the hospital because he was sick enough, but was sent back to have a diagnosis made, because the signs and symptoms of one kind or another had persisted and kept recurring. This swelling in the back of his hand had now appeared.

Since he has been in the hospital this second time there has been a good deal of discussion as what the diagnosis was. A good many

held to the idea that there was some tuberculosis, probably some pulmonary tuberculosis coming to the surface and causing this pleural inflammation. Everybody knew that there were some signs of cardiac lesion present, though there was very little sign of embarrassment in any sense to the circulation. Then with this evidence of a low-grade inflammatory process in the wrist and elbow, in the pleuræ on both sides, and down over his liver, it was suggested that he might have a low-grade sort of infection which was very extensive and expressed itself as effusion into the various cavities—the serous cavities, such as the pleural cavity, the pericardial cavity, and the peritoneum, which could be grouped together under the general term polyserositis, which is an unsatisfactory term unless you know what it means—inflammation of many serous cavities. That was first described in America by Doctor Kelley, under the term "multiple serositis," in the 1903 number, Volume cxxv, of the *American Journal of the Medical Sciences.* He did not refer to this particular type of case, but referred to a group of cases in which you have adherent pericardium and recurring ascites, so that the patients sometimes have their abdomens tapped a great many times, and either pleural effusion or pleural adhesions, and with that a perihepatitis and perisplenitis; and the peculiarity of the perihepatitis and perisplenitis was that there was over the surface of the liver and spleen a sort of callus-like formation, rather white areas, very dense, and when you cut them they had practically no blood in them, varying in thickness from 1 to 2 mm. up to .5 cm., and really looking like the cut surface of a callus on the hand of a laboring man. They were scattered irregularly over the liver, and had been described in the past as resembling frosting on cake or icing, having a somewhat glassy or ice-coating appearance. They were originally described by Germans, who spoke of it as "zuckergussleber," or pouring sugar over the surface of the liver. That was described by Pick, a German, and it is sometimes spoken of as Pick's disease. It was also described by an Italian, Concato, and is sometimes spoken of as his disease. After Kelley, in this country, described the disease there were many other reports of individual cases and groups of cases, and the idea of polyserositis rather spread out from this pretty definite symptom complex to include a larger group in which there were constantly recurring or continued effusions into serous cavities with a consider-

able amount of exudate in most of the cases; sometimes ascites, sometimes ascites and pleural effusion, sometimes pericardial effusion, pleural effusion and ascites, and in any of those cavities instead of effusion adhesions and varying clinical pictures on that basis. The etiology in most of those cases is not known, because the fluid in most of those cases is negative for tubercle bacilli, and animal examination gives no evidence of tuberculosis. It is some kind of low-grade inflammatory process.

We have this patient, who has been demonstrated to have some sort of effusion in his pericardial cavity and some sort of effusion in the serous surfaces about the joints and tendon sheaths. The other possible thing that lurked in our minds when he was under observation before, and never entirely left, was that there was some sort of tuberculous process, and this is probably a tuberculous pericardial effusion. We get a certain number of cases with chronic pericarditis with effusion that develop insidiously, as in this patient; the fluid accumulates to a very considerable extent, so that you get large amounts of fluid accumulating very gradually, so that the pericardium stretches, and this usually does not interfere much with the circulation, so they probably do not have cardiac embarrassment, but have other of the symptoms, and the underlying cause is thought to be tuberculosis. In our animal examination we may find tubercle bacilli. If we find no tubercle bacilli we have simply got to say that this is some kind of low-grade inflammation, and it is as well to call it polyserositis as it is to say it belongs to any other symptom complex.

It illustrates very well the difficulty in diagnosis in many of these cases. It is difficult to make out effusion in the pericardium. These two plates are very much like the X-ray plates that I showed you on the patient named Zellaway, who came to autopsy and had a big globular heart, due to chronic myocarditis. We have other plates on patients giving very much the same picture, and there is no pericardial effusion, but there is an enlarged heart, a marked degree of pericardial thickening with adhesions, sometimes with complete obliteration of the pericardial sac—adhesive pericarditis—so that it is very difficult in one of those cases, until the fluid becomes very marked, to differentiate between pericardial effusion, adhesive pericardium with an enlarged heart, and dilatation and hypertrophy of

the heart without any pericarditis. The especially helpful part, in so far as the X-ray is concerned, is the filling out of the upper part of the pericardial sac, a good deal of filling out of the angle between the right border of the heart and liver—change from an acute angle to a right angle and then to an obtuse angle—though this is usually made out better by percussion than X-ray, because usually in the X-ray it remains acute, even when fluid is present; as far as physical examination is concerned, there is the large size of the heart without cardiac embarrassment, with the absence of much pulsation. There is very little to be felt in the way of an impulse when you palpate the heart, but here is a big heart so far as the outline of dulness is concerned, and the heart is not very much embarrassed, so there is no reason why you should not get a cardiac impulse if the heart is doing its work well and is large unless there is something between the contracting muscle and your hand. That something may be fluid or may be a thickened pericardium. When you can feel the impulse inside of the border of dulness there is every probability of that difference between the point of impulse and the point of cardiac dulness—the outer border—being fluid rather than thickened pericardium, though sometimes it is thickened pericardium. The exploratory needle is resorted to from the point of view of diagnosis and resorted to from the point of view of treatment of the case. If this 600 Cc. of fluid, instead of accumulating slowly, had accumulated very much more rapidly, the patient would have had signs of cardiac decompensation, the pulse would have become rapid, the patient would probably have gotten cyanotic, and if the fluid was not drawn off he would have died with circulatory failure. This picture, where it accumulates gradually, does not have circulatory failure. When there are adhesions between the two layers of the pericardium there are apt to be, also, adhesions between the pericardium and diaphragm, and then the heart contracting pulls on the ribs and you can make out a Broadbent sign. Not infrequently we have some cases of pericarditis with no adhesions between the pericardium and the diaphragm and lungs, and these do not give a Broadbent sign, so the absence of this sign does not rule out the presence of adherent pericardium. Finding it is rather suggestive of adherent pericardium.

The fluid will probably reaccumulate, particularly if it is a tuber-

culous process it probably will recur, and the patient will have to be tapped again. Some of the cases have been tapped a good many times. In other cases with one tapping the fluid disappears and does not reappear like pleural effusion, and the patient gets well. I will report later as to what happens as far as the progress of the case is concerned.

January 22, 1918: You remember the patient that I showed you two months ago with pericardial effusion in which the etiology was not clear (Medical, No. 7527). The patient had had pleurisy without effusion, pericardial effusion, and various joints and tendon sheaths were involved in an inflammatory process. That patient had his pericardium tapped twice. When I showed him it was after taking out 690 Cc. of fluid, and recently 480 Cc. of fluid was taken out. He has secondary anæmia now and has been transfused several times. Just recently he had his tonsils taken out. They were found to be in bad condition, containing pus pockets. Notwithstanding the fact that he had tonsillitis only twice, eighteen years ago, and had not had tonsillitis since, the findings rather suggest, as far as they have gone, that in that particular the pericarditis is related to the tonsillitis and possibly is a rheumatic thing. The fluid was put into a guinea pig at the second tapping. In the case of the first fluid the guinea pig either got lost or died immediately after the injection. It did not go along far enough to give any results as far as tuberculosis is concerned.

March 22, 1918: Pericardial fluid obtained on January 11 gave a guinea pig test negative for tuberculosis. On December 27 the patient had a right peritonsillar abscess incised. On January 17 he was transferred to the surgical service, and on January 18 tonsillectomy was performed and chronically inflamed tonsils were found. On January 20 he was returned to the medical service, and for a considerable time had a painful inflamed throat. He continued to run an irregular temperature. Then his throat became inflamed again, exudate developed, and on February 27 a positive culture of bacillus diphtheriæ was obtained from his throat, and, though the membrane did not look typical of diphtheria, he received a considerable amount of antitoxin, which had but little effect on the general appearance of his throat. His anæmia became more marked and he received several transfusions. He gradually lost

ground. A great deal of necrosis developed in the region of his tonsil. He finally had several hemorrhages from this necrotic material, and died on March 17. Autopsy showed an extensive area of necrosis and slough in his pharynx and tongue, localized pericardial adhesions with only a moderate amount of fluid in his pericardium, areas of thickening of the pleura and evidences of fibrous adhesions and tags between the liver and the diaphragm. No evidence of tuberculosis was found.